



Missouri Department of Health and Senior Services

P.O. Box 570, Jefferson City, MO 65102-0570 Phone: 573-751-6400 FAX: 573-751-6010
RELAY MISSOURI for Hearing and Speech Impaired 1-800-735-2966 VOICE 1-800-735-2466

Peter Lyskowski
Director



Jeremiah W. (Jay) Nixon
Governor

December 21, 2016

John E. Price
Carnahan, Evans, Cantwell & Brown, P.C.
2805 S. Ingram Mill Road
P.O. Box 10009
Springfield, MO 65808

RE: Fantastic Caverns Employee Notification of TCE Exposures



Superfund

Dear Mr. Price:

0400

12/21/16

55

The Missouri Department of Health and Senior Services (MDHSS) recently received a copy of the enclosed report prepared by Ozark Underground Laboratory (OUL), *Fantastic Caverns Information for Employees on TCE in Cave Air*, dated September 1, 2016, that was reportedly presented to employees of your client, Fantastic Caverns, on October 13, 2016. While MDHSS appreciates that Fantastic Caverns is sharing information with employees, the information provided in this report is very troubling because it misinforms the employees of trichloroethylene (TCE) exposure concerns and potential health risks. The U.S. Environmental Protection Agency (EPA) also expressed similar concerns about the information provided to Fantastic Cavern's employees in a recent letter (enclosed) to the Missouri Department of Natural Resources (MDNR). MDNR forwarded that letter to us and indicated that they share EPA's concerns. Therefore, please consider this letter a request by our combined agencies that employees of Fantastic Caverns be immediately provided accurate health information on TCE exposure.

Of specific concern in the OUL report is the comparison of the cave air TCE levels to the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) and the conclusion that, since the levels detected in cave air are below this OSHA level, there is no health risk to employees. While OSHA does in fact regulate workplace health and safety, OSHA PELs apply to commercial/industrial operations where the subject chemicals are in active use or production. Since the source of TCE in air within Fantastic Caverns is from environmental contamination and not from the use or production of TCE at the facility, OSHA standards are not applicable for the cave. MDNR previously provided information to the cave owner by email on September 2, 2016 regarding the inappropriateness of use of OSHA PELs at this site, and that information sheet is enclosed again with this letter for reference. In addition to the information provided in this enclosure, it is also worthy to note that OSHA too recognizes that many PELs "are outdated and inadequate for ensuring protection of worker health." (www.osha.gov/dsg/annotated-pels)

EPA uses current science to calculate health protective standards for chemical exposure to protect individuals, including workers, at sites with environmental contamination. An EPA Region VII memo titled *Action Levels for Trichloroethylene in Air* (November 02, 2016) provides the recommended action

www.health.mo.gov

Healthy Missourians for life.

The Missouri Department of Health and Senior Services will be the leader in promoting, protecting and partnering for health.

AN EQUAL OPPORTUNITY / AFFIRMATIVE ACTION EMPLOYER: Services provided on a nondiscriminatory basis.

levels for TCE in air as well as information on characterizing and addressing human health risk from less than life-time exposures. The memo is included as an enclosure.

Air samples from the cave collected as long ago as 2004 and as recently as this year have shown intermittent elevations of TCE significantly above the current EPA level. Both MDHSS and MDNR previously recommended that employees of Fantastic Caverns be informed that TCE has been detected in cave air above levels of health concern and that the employees be provided with applicable health information on TCE exposure. The agencies followed up on several occasions (September 2, 19, and October 6, 2016) by providing the enclosed employee fact sheet to assist with informing employees. It is imperative that employees are adequately informed of the TCE exposure concern and the potential health risks. Therefore, employees must be provided with the information in the enclosed Fantastic Caverns Employee Fact Sheet (developed by MDHSS and MDNR) and the TCE fact sheet (developed by the federal Agency for Toxic Substances and Disease Registry). Because of the potential risk to human health from elevated TCE exposures, please provide MDHSS with documentation that owners and employees at Fantastic Caverns have received this important information regarding health risks from exposure to TCE.

Furthermore, the workers should be made aware that MDHSS is available to answer health-related questions as needed. MDHSS is available upon request to meet in person, provide a presentation, and address any health concerns that Fantastic Caverns' owners or employees may have.

If you have any questions or concerns regarding these issues, please feel free to contact me at (573) 751-6102. Additionally, if you have any questions for MDNR, please contact Dennis Stinson at (573) 751-1388.

Sincerely,



Jonathan Garoutte, Chief
Bureau of Environmental Epidemiology

JG:DW:MDH

cc: Dennis Stinson, MDNR
Steve Sturgess, MDNR
Mary Peterson, EPA
Spencer Williams, ATSDR

Enclosures



Staff Meeting-TCE in cave air

October 13 th 2016	signature
Kirk Hansen	<i>[Signature]</i>
Scot Franson	<i>[Signature]</i>
Kevin Pratt	<i>[Signature]</i>
Madison Campbell	<i>[Signature]</i>
Marci Riedle	<i>[Signature]</i>
Jill Joy	<i>[Signature]</i>
Susan Pagel	<i>[Signature]</i>
Scarlet Casey	<i>[Signature]</i>
Rick Butler	<i>[Signature]</i>
Nicole Boucher	<i>[Signature]</i>
Jared Bruegman	<i>[Signature]</i>
Walter Rice	<i>[Signature]</i>
Martin Garrison	<i>[Signature]</i>
Charles Warner	<i>[Signature]</i>
Richard Brake	<i>[Signature]</i>
Victor Hyatt	<i>[Signature]</i>
Ronald Baas	<i>[Signature]</i>
Karen Solomon	<i>[Signature]</i>
Amanda Hardin	<i>[Signature]</i>
Melissa Schmitz	<i>[Signature]</i>
Hunter Slack	<i>[Signature]</i>
Katherine Altie	<i>[Signature]</i>
Michael Moore	<i>[Signature]</i>
Misty Lane	<i>[Signature]</i>
Russell Stephenson	<i>[Signature]</i>

Craig Miller

[Signature]

Fantastic Caverns Information for Employees on TCE in Cave Air

**Tom Aley, Missouri Registered Geologist #0989
Senior Hydrogeologist and President
Ozark Underground Laboratory
September 1, 2016**

Purpose

Very small concentrations of the chemical trichloroethylene (TCE) can sometimes be detected in the air of Fantastic Caverns. Management of the Caverns asked the Ozark Underground Laboratory to prepare written information for employees so that they can be informed about the issue. That is the purpose of this brief report.

TCE has, so far as we can determine, never been used in the cave or at any point on the cave property where it might enter the cave. It enters the cave either through air or water. It is a volatile compound and can move from water into air or from air into water. The TCE in the cave air is attributable to spillage, disposal, or the underground movement of this compound within the 12.72 square mile recharge area for the cave.

At concentrations much higher than those encountered in Fantastic Caverns TCE is identified by the government as a carcinogen (cancer-causing agent). Some very limited studies have suggested that low concentrations of TCE in air might present some risk of health consequences. TCE in either air or water could possibly adversely impact cave features.

Background

Trichloroethylene (TCE) was heavily used as a solvent from the early 1920s through the 1970s, but after the 1970s it became less popular due to environmental concerns (Bakke et al. 2007). TCE is recognized as an occupational carcinogen in water and air. Some studies have suggested an association between TCE exposure in older women and heart defects in their babies but study results are questionable because of the small numbers of cases, insufficient exposure characterization, chemical co-exposures, and other methodological deficiencies (Chiu et al. 2013). One of the likely, but unassessed, co-exposures is to Zofran. This drug was not approved by the FDA for treatment of morning sickness, but has been given to about a million women a year for this condition (DrugWatch, accessed 9/1/16). There is a class action suit against the maker of this drug claiming that, among several effects, it produces heart defects in babies born to mothers who took the drug during pregnancy (DrugWatch, accessed 9/1/16).

Exposure to TCE in workplace air is regulated by OSHA. The limit in air is 100 parts per million (ppm) of TCE time-weighted average over an 8-hour work period. This level is defined (as per ATSDR 2007) as "concentration for a normal 8-hour workday and 40-hour work week

[that is] set at a level at which nearly all workers may be repeatedly exposed without adverse effects." The 100 ppm concentration is equal to 537,000 micrograms of TCE per cubic meter of air. There is no other established federal standard although the US Environmental Protection Agency (EPA) has a current target concentration in air impacted by Superfund sites of 6 micrograms of TCE per cubic meter of air. No, this is not a typo. Yes, the legal limit established by OSHA is over 90,000 times greater than the EPA target. There is no scientific reason that TCE derived from a Superfund site should be 90,000 times more harmful than TCE from other sources. The appropriateness and reasonableness of this target is not supported by credible data and is strongly challenged (Coffin et al. undated). Also, there is no Superfund site in the recharge area for Fantastic Caverns.

Bakke et al. (2007) report that the mean TCE concentration in workplace air at sites where TCE is used is 38.2 ppm (208,954 micrograms per cubic meter). This value is based on all industries and decades, but most measurements are from the 1950s, 1970s, and 1980s. An estimate by National Institute for Occupational Safety and Health (NIOSH) in 1973 was that 200,000 workers were potentially exposed to TCE. In 1978 the number was revised downward to 100,000 fulltime exposures with up to 3.5 million more workers exposed to continuous low levels or to brief exposures to various levels of TCE (Sittig 1985). I have found no studies suggesting that children of workers who had exposures to TCE many thousands of times greater than the EPA target concentration were born with an elevated frequency of deformed hearts. Perhaps such studies exist; if they do not their absence suggests that obvious investigations have been omitted in EPA's rush to establish minute target concentrations.

Current production of TCE in the US exceeds a million pounds per year. It can be purchased without any permit or training and is easily available on the internet. Its many uses have included:

- As a dry cleaning liquid.
- Metal degreasing and in cleaning electronic parts. This is the largest single use.
- Dissolving fats, greases, and waxes. It has sometimes been used in cleaning sewers and septic systems of fat and grease. It undoubtedly works wonders on clogged sewers or grease traps at cafés!!
- Cleaning auto parts, and especially for cleaning carburetors and brakes.
- For removing caffeine from coffee and other oils from spices.
- As a refrigerant and heat exchange liquid.
- As a fumigant.
- In medicine as an anesthetic where it was often administered during child birth.

Because of the many different uses of TCE at multiple sites the compound is widely dispersed in both air and water. Verschueren (1983) reports that TCE is found in municipal sewer air at 10 to 100 parts per million (ppm). This equals a concentration range in sewer air of 54,700 to 547,000 micrograms per cubic meter of air. The TCE in sewer air is derived from disposal of the compound into drains as a liquid. Within the sewers some of the TCE volatilizes and is then present in both the air and the water of the sewers. TCE can move from water into

air or from air into water and, as a result, in karst areas can be transported underground by either (or both) air and water. The concentration of TCE in municipal sewer air today is probably smaller than reported by Verschueren (1983), yet sewers (and especially older ones) are notorious for leaking sewage (and thus TCE) into groundwater in karst areas. There are tens of miles of private and public sewers in the 12.71 square mile recharge area for Fantastic Caverns. The sewers are in addition to the dozens of point sources in the recharge area where TCE has been dumped or spilled into the karst groundwater system.

There are three factors that explain the long-term persistence of TCE in the subsurface. First, TCE is not very soluble in water. To dissolve one pound of TCE in water you would need 1,280 pounds of water and good mixing. Second, TCE is 1.46 times denser than water. Where water is detained in cavities in limestone the undissolved TCE sinks to the bottom of the detaining pools and is not flushed out by stormflows or by becoming rapidly dissolved in the passing water. Third, and this is especially true in Missouri karst areas, hazardous waste sites with TCE are frequently inadequately managed. It is common to find that TCE removal from the subsurface has been incomplete and that off-site migration control has been grossly ineffective.

TCE at Fantastic Caverns

Potential Sources

First, there is no indication that TCE has ever been used inside Fantastic Caverns. Any TCE in the cave's air or water is from off-site sources.

The area that contributes water to Fantastic Caverns (called the Fantastic/Big Williams Spring Recharge Area) was delineated by Aley and Thomson (2002) based on all tracing done up to that date by all entities including MDNR and Springfield City Utilities. The data base included 210 traces from 1898 to 2002 in Greene County. The recharge area encompasses 12.71 square miles.

There are many potential source areas for TCE in this large recharge area. One site that has received substantial attention as a TCE source in Greene County is the former Litton wastewater lagoon. It was located in a sinkhole east of the Springfield Regional Airport. MDNR introduced dye into this sinkhole on January 19, 1989 (Aley and Thomson 2002). Dye from this introduction was NOT detected at either Big Williams or Fantastic Caverns Springs. Instead, the dye from this introduction was detected at Ritter Springs West. The dye introduction for the MDNR trace was east of the recharge area divide between Ritter Springs and the Fantastic/Big Williams Springs Complex as drawn by Aley and Thomson (2002).

The former Litton plant is located west of the Litton wastewater lagoon and is within the delineated Fantastic/Big Williams Spring Complex. It is possible that there may be TCE in groundwater in this area but as of the time of the Aley and Thomson (2002) study there had been no groundwater tracing from this plant site to any hydrologically associated springs. There has been a dye trace conducted from a sinkhole at the airport that is about 1,800 feet

northwest of the former Litton lagoon. Dye from that trace discharged at the Fantastic Caverns/Big Williams Spring Complex.

The former Litton facility is about 17,000 feet from Fantastic Caverns. I am not aware of any data to support attributing TCE in Fantastic Caverns to the former Litton plant. Given the large recharge area for Fantastic Caverns and past and current land use in the area there are many potential sources for the TCE detected in the cave. The former Litton lagoon is not among them.

TCE Measurements

Monitoring for TCE has been conducted in Fantastic Caverns by MDNR. This has occurred on five dates with a total of 10 measurements. The values are shown below.

Season	Number of Measurements	Range of Values (micrograms/cubic meter)	Mean (micrograms/cubic meter)
Winter	5	<5.3 to 53	11
Spring	3	0.94 to 150	51
Summer	2	61 to 88	75
Fall	0		
Annual	10	0.94 to 150	35

Note: In calculating means values, those shown as less than were assumed to be zero.

Based on the measurements that have been made I conclude that:

- The OSHA limit for TCE in workplace air is 547,000 micrograms per cubic meter. This is over 15,000 times higher than the mean value measured in Fantastic Caverns.
- The highest value measured in the cave was 150 micrograms per cubic meter. The OSHA limit is over 3,600 times higher than this measurement.
- The OSHA limit is for an 8-hour time-weighted average. If a person spends four hours per day in the cave the OSHA limit would be over 30,000 times higher than the actual employee exposure based on the mean measured value and it would be about 7,300 times higher than the actual employee exposure at the largest concentration measured.
- Because of airflow patterns, the portions of the cave likely to have the highest TCE concentrations are the locations measured. Average TCE concentrations in the cave are likely to be about half of the measured concentrations. As a result, employee exposures to the air in Fantastic Caverns are minute.
- The MDNR monitoring has shown that Fantastic Caverns is fully in compliance with the OSHA limits for TCE in workplace air. No more monitoring is needed or is appropriate.

Impacts to the Cave

An air contaminant such as TCE has the potential to impact the nature and extent of calcite speleothems (cave formations). The technical literature, for example Hill and Forti

(1997), is basically speculative on this topic. Regardless of this, TCE in the cave air is undesirable but will undoubtedly persist for many years. Its impact on cave organisms at the concentrations likely found in the cave stream is also unknown.

One privately-owned show cave has recently installed an elaborate forced air ventilation system and other features to reduce TCE concentrations in cave air to levels below the EPA target level of 6 micrograms per cubic meter of air. This will almost certainly cause environmental damage to the cave; the severity of the damage to the cave and to the show cave business has not been determined. This other show cave is known to be connected to a distant Superfund site and in this way it differs from Fantastic Caverns. The EPA has done no environmental assessment of the impact of this dramatic alteration to the natural microclimate of this other show cave.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Trichloroethylene toxicity; what are the U.S. standards for trichloroethylene exposure? Environmental health and medicine education. <http://www.atsdr.cdc.gov/csem/csem.asp?csem=15&po=8>. 4p.

Aley, Thomas and Kenneth Thomson. 2002. A study of septic field performance and recharge area delineation for 12 spring systems in Greene County, Missouri. Ozark Underground Laboratory contract report to Greene County, Missouri. 79p + 3 large maps.

Bakke, B.; P.A. Stewart; and M.A. Waters. 2007. Uses of and exposure to trichloroethylene in U.S. industry: a systematic literature review. Jour. Occupational Environmental Hygiene Vol. 4:5, pp. 375-390.

Chiu, Weihsueh A. plus 15 others. 2013. Human health effects of trichloroethylene: key findings and scientific issues. Environmental Health Perspectives Vol. 121:3, pp. 303-311.

Coffin, Richard C.; Ron Morgan; Gulhuly; and Jan Tom Boert. Undated. EPA's concern over TCE vapor intrusion is misguided. Law360 on-line newsletter. 6p.

DrugWatch. <http://www.drugwatch.com/zofran/lawsuits/>. Accessed September 1, 2016.

Hill, Carol and Paolo Forti. 1997. Cave minerals of the world. Second Edition. National Speleological Society. 463p.

Sittig, Marshall. 1985. Handbook of toxic and hazardous chemicals and carcinogens, Second Edition. Noyes Publications. Pp. 883-885.

Verschuieren, Karel. 1983. Handbook of environmental data on organic chemicals, Second Edition. Van Nostrand Reinhold Co. Pp. 1131 to 1136.

Waters, E.M.; H.B. Gerstner; and J.E. Huff. 1977. Trichloroethylene. I. An overview. Jour Toxicology and Environmental Health Vol. 2:3, pp. 671-707.

S:\tom\tce13.doc



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 7

11201 Renner Boulevard
Lenexa, Kansas 66219

DEC 09 2016

Steve Sturgess
Missouri Department of Natural Resources
P.O. Box 176
Jefferson City, Missouri 65102-0176

Dear Mr Sturgess:


Thank you for providing a copy of the Fantastic Caverns Information for Employees on TCE in Cave Air, authored by Tom Aley, dated September 1, that was provided to employees by Fantastic Caverns at a facility meeting on October 13, 2016 (TCE Fact Sheet). The U.S. Environmental Protection Agency considers it an important step that the employees of Fantastic Caverns have information about the measured levels of TCE at the caverns. However, the EPA believes there is information in the TCE Fact Sheet that may mislead employees about the risks of exposure to TCE in air. Specifically, the EPA action level is based on the most current scientific studies that were not available when the Occupational Safety and Health Administration established the Permissible Exposure Levels for TCE. As noted on OSHA's website (www.osha.gov/dsg/annotated-pels) many PELs "are outdated and inadequate for ensuring protection of worker health." The TCE Fact Sheet also incorrectly dismisses the need to study or address the TCE contamination to ensure the health of employees and guest tourists.

The TCE risk-based action level for a typical industrial/commercial scenario with an 8 hour workday is $6 \mu\text{g}/\text{m}^3$ (EPA, 2016). This level is based on the TCE chronic reference concentration of $2 \mu\text{g}/\text{m}^3$ and an assumption that a single exposure to TCE at any time during an approximate three-week period in early pregnancy could result in one or more types of cardiac malformations. Thus, the critical exposure period of concern used to evaluate the potential for heart defects and derive the TCE action level is one day. Chronic exposure to TCE may affect the central nervous system, kidneys, liver, immune system, and/or male reproductive system, and poses cancer risks. An exceedance of the TCE action level indicates a potential imminent threat to human health. The EPA applies this analysis to sites with environmental contamination, and the OSHA standard referred to in the employee fact sheet is not a risk-based level that the EPA would apply to this situation.

The employees of Fantastic Caverns should be made aware how the EPA calculates the risks from exposure to TCE, and how the EPA uses the most current scientific information to protect workers and others at sites with environmental contamination. The EPA is available to discuss with MDNR and MDHSS options for enhancing communication with employees at Fantastic Caverns to ensure they receive correct information regarding potential health risks from exposure to TCE.

If you have any questions, please call me at (913) 551-7882.

Sincerely,



Mary P. Peterson, Director
Superfund Division



Printed on Recycled Paper

OSHA TCE LEVELS

The OSHA PEL for TCE is 100 ppm, which equates to 537,423 $\mu\text{g}/\text{m}^3$.

While OSHA does in fact regulate workplace health and safety, OSHA workplace air standards are applicable to commercial/industrial operations where the subject chemicals are in active use or production. Since the source of TCE in air within Fantastic Caverns is from environmental contamination and not from the use or production of TCE, OSHA standards are not applicable in this instance.

Where potential worker exposures are due to contamination of the environment and not from work practices, risk-based levels protective of both cancer and non-cancer effects (including sensitive individuals) are applicable to both assessing potential exposures and developing risk-based remediation levels.

The key distinctions between OSHA standards and risk-based levels include the underlying assumptions about the context of workplace exposures, the characteristics of the workers being protected, and the level of protection afforded to workers.

OSHA standards specifically apply to workplaces where workers are exposed to hazardous chemicals used in or generated as a result of routine work activities. These workers are assumed to be aware of the chemicals to which they are exposed and can obtain information on them through Right-to-Know laws. OSHA standards assume not only that workers are knowingly exposed to specific chemicals in the workplace, but that they also receive additional protection and training to mitigate exposures. OSHA requires workers to be trained to control or prevent exceedances of its exposure standards (including the use of personal protective equipment to help prevent excessive exposures), and also requires periodic worker health monitoring to ensure that excessive exposures are not occurring. In addition, OSHA standards were developed assuming a healthy adult worker and were not necessarily developed to be protective for sensitive individuals, who may have preexisting health conditions, etc.

Although, MDHSS is not specifically questioning the protectiveness of the OSHA levels, we would like to point out that these levels have not been updated in decades and are not based on current science and toxicity data. Scientific evidence shows TCE is more toxic than previously thought and, thus, there have been recent updates to toxicity values used in assessing exposure and developing risk-based remediation levels. Currently, there are several orders of magnitude difference between OSHA standards and current risk-based levels for TCE.

For additional information or questions, you may contact Michelle Hartman of Missouri Department of Health and Senior Services at 573-751-6102.



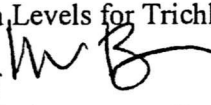
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 7

11201 Renner Boulevard
Lenexa, Kansas 66219

NOV 02 2016

MEMORANDUM

SUBJECT: EPA Region 7 Action Levels for Trichloroethylene in Air

FROM: Mike Beringer, Chief 
Environmental Data & Assessment Branch
Environmental Sciences & Technology Division

TO: Branch Chiefs
Waste Enforcement and Materials Management Branch
and Waste Remediation & Permitting Branch
Air and Waste Management Division

Branch Chiefs
Superfund Division

The purpose of this memorandum is to update the U.S. Environmental Protection Agency Region 7 RCRA and Superfund programs on the recommended action levels for trichloroethylene (TCE) in air, and provide information on characterizing and addressing human health risks from less-than-lifetime exposures. The action level for a residential scenario is $2 \mu\text{g}/\text{m}^3$, and the action level for an industrial/commercial scenario with an 8-hr workday is $6 \mu\text{g}/\text{m}^3$. Equations to allow derivation of action levels for alternative scenarios, such as a 10-hr workday, are presented. As described in this attachment, it is assumed that an exposure to TCE at any time during an approximate three-week period in early pregnancy could result in one or more types of cardiac malformations. Thus, the critical exposure period of concern used to evaluate the potential for heart defects and derive action levels for TCE is one day. An exceedance of the TCE action level indicates a potential imminent threat to human health. Region 7 should expedite early or interim action(s) to eliminate, reduce, and/or control the hazards posed by the site as quickly as possible. If you or your staff have any questions or need further assistance, please contact Kelly Schumacher (x7963).

EPA Region 7 Action Levels for Trichloroethylene in Air.	
<i>Exposure Scenario</i>	<i>Action Level</i>
Residential (24 hours/day)	$2 \mu\text{g}/\text{m}^3$
Industrial/Commercial (8 hours/day)¹	$6 \mu\text{g}/\text{m}^3$

¹ Site-specific action levels should be derived when the workday differs from 8 hours/day.

RECEIVED

Attachment

NOV 03 2016

AWMD/WRAP IC



Printed on Recycled Paper

EPA Region 7 Action Levels for Trichloroethylene in Air

Introduction

In 2011, the latest human health toxicity values for trichloroethylene were published by the United States Environmental Protection Agency's Integrated Risk Information System program (EPA, 2011a). As discussed in this document, these new values are partly based on developmental health effects that result from less-than-lifetime exposures. In contrast, the toxicity values typically used to evaluate potential health risks and derive action levels at Superfund and RCRA sites are based on health effects associated with long-term, or chronic exposures. Further, the equations and exposure parameters used typically reflect all or a significant portion of a person's lifetime. Once the current TCE values were released, the protectiveness of using traditional approaches to assess and address TCE exposures was questioned. The purpose of this memorandum is to update the EPA Region 7 RCRA and Superfund programs on the recommended action levels for TCE in air and provide information on characterizing and addressing human health risks from less-than-lifetime exposures. To support these objectives, the window of susceptibility for the developmental toxicity associated with TCE is examined, the critical exposure period of concern is identified, and the appropriate exposure parameters and equations are elucidated.

Toxicity Assessment

The EPA's final toxicological review by the IRIS program incorporates comments by the U.S. National Academy of Sciences (National Research Council, 2006), two U.S. EPA Science Advisory Boards (EPA, 2002 and 2011b), the Executive Office of the President (Office of Management and Budget, 2009 and 2011), the U.S. Department of Defense (DOD, 2009a, 2009b and 2011), the National Aeronautics and Space Administration (NASA, 2009 and 2011), internal Agency reviewers, and the public, among others. The Halogenated Solvents Industry Alliance, Inc., which represents the interests of TCE manufacturers and producers, submitted a Request for Correction of the TCE IRIS assessment (HSIA, 2013), which was denied by the EPA's Acting Assistant Administrator (EPA, 2015). The HSIA then submitted a Request for Reconsideration (HSIA, 2015), which was also denied by the EPA (EPA, 2016a). The EPA found the Requests "directly contrary to the SAB's conclusions and recommendations, such that to accept HSIA's RFC/RFR would require EPA to reject SAB's advice" (EPA, 2016a).

The EPA's Office of Land and Emergency Management recognizes an IRIS assessment as the official Agency scientific position regarding the toxicity of a chemical based on the data available at the time of the review (EPA, 2003). As such, IRIS is generally the preferred source of human health toxicity values used to evaluate risks at Superfund and RCRA hazardous waste sites. In accordance with Directive 9285.7-53 (EPA, 2003), the 2011 IRIS TCE toxicity values will be used to evaluate risks and derive action levels by the Region 7 RCRA and Superfund programs until the 2011 values are either revised or rescinded.

Non-Carcinogenic Health Effects

In general, the EPA assumes that a dose or exposure level exists below which adverse non-carcinogenic health effects will not occur (EPA, 1989). Below this threshold, it is believed that exposure to a chemical is tolerated without adverse effects. Adverse health effects occur only when physiologic protective mechanisms are overcome by exposure to doses or concentrations above the threshold. For chronic toxicity values, the first adverse effect (or its known precursor) that occurs to the most sensitive species as the dose rate of an agent increases, regardless of the exposure duration, is designated the

critical endpoint. The dose or exposure at which the critical endpoint is observed is the point of departure. Uncertainty factors, ranging from 1 to 3,000, reflecting limitations of the data used are applied to the point of departure to derive the inhalation reference concentration. The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 1989).

The 2011 Scientific Advisory Board panel recommended that, “The two endpoints for immune effects from Keil *et al.* (2009) and the cardiac malformations from Johnson *et al.* (2003) should be considered the principal studies supporting the RfC” (EPA, 2011b). The panel considered the immune effects and cardiac malformations co-critical endpoints (EPA, 2011b). In accordance with the SAB panel recommendations, the IRIS program based the TCE chronic reference concentration of $2 \mu\text{g}/\text{m}^3$ on these two co-critical endpoints, each of which can support the RfC independently: autoimmune disease following chronic exposure in adults (0.00033 ppm , or $1.8 \mu\text{g}/\text{m}^3$) and heart defects following exposure during early pregnancy (0.00037 ppm , or $2.0 \mu\text{g}/\text{m}^3$). The RfC is also supported by nephrotoxicity (kidney effects) following chronic exposure in adults (0.00056 ppm , or $3.0 \mu\text{g}/\text{m}^3$). Following publication of these values, the developmental cardiac effects were further addressed by the IRIS program in “TCE Developmental Cardiac Toxicity Assessment Update” (EPA, 2014a) and by scientists in the EPA’s Office of Research and Development in the peer-reviewed literature (Makris *et al.*, 2016).

Chronic exposure to TCE poses a potential human health hazard to the central nervous system, kidneys, liver, immune system, and male reproductive system. As mentioned above, immunotoxicity in adults is considered a co-critical endpoint, at a slightly lower concentration than that associated with cardiac defects. Overall, the IRIS program concluded that “the human and animal studies of TCE and immune-related effects provide strong evidence for a role of TCE in autoimmune disease and in a specific type of generalized hypersensitivity syndrome” (EPA, 2011a). Kidney toxicity was considered a supporting endpoint, with high confidence found in multiple lines of evidence in both human and animal studies.

Short-term exposures to TCE during pregnancy are associated with many forms of developmental toxicity, including spontaneous abortions, decreased growth, developmental neurotoxicity, developmental immunotoxicity, and birth defects. However, the critical developmental endpoint is cardiac malformations. The primary types of heart defects observed with TCE exposures include atrial and ventricular septal defects, which are holes in the wall (septa) between the top two chambers (atria) or bottom two chambers (ventricles) of the heart, and pulmonary and aortic valve stenoses, which are thickened or fused heart valves that do not properly open and/or close and may leak blood. The critical window of susceptibility for these types of defects is an approximate three week period (i.e., valvuloseptal morphogenesis, or the period in which major cardiac morphogenic events such as heart valve formation occur) approximately four to seven weeks after conception, early in the first trimester of human pregnancy (Dhanantwari *et al.*, 2009). The type and severity of the resulting cardiac malformation or malformations depends on the timing and level of exposure to TCE within this approximate three week period. Exposures that clear the body before this period do not impact the heart valves and septa, because they have not yet begun to form. In humans, TCE and most of its metabolites are eliminated within a week of exposure (EPA, 2011a).

Carcinogenic Effects

The EPA evaluates carcinogenicity in two parts (EPA, 2005a). First, the Agency evaluates all available scientific information and assigns a weight-of-evidence classification based on a compound’s potential to cause cancer in humans. In the absence of sufficient data regarding the mode of action or if the

weight-of-evidence supports a mutagenic mode of action, the EPA generally assumes that any exposure to a chemical will increase an individual's risk of developing cancer. Under this default approach, there is no threshold below which the probability of developing cancer is zero. Second, a toxicity value is derived to define the quantitative relationship between dose or concentration and carcinogenic response. For inhalation exposures using the default approach, this value is known as the inhalation unit risk. The IUR is a generally plausible upper-bound estimate of the increased probability of developing cancer following a lifetime of exposure. This value is used to estimate the increased risk of developing cancer from inhalation of potentially carcinogenic chemicals.

Following the EPA's Guidelines for Carcinogen Risk Assessment (EPA, 2005a), the IRIS program has evaluated the carcinogenic potential of TCE and has classified it as "carcinogenic to humans" by all routes of exposure. This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer, strong evidence of non-Hodgkin's lymphoma, and more limited evidence of liver and biliary tract cancer. The inhalation unit risk for TCE, based on these combined cancer types, is $4.1\text{E-}06\ (\mu\text{g}/\text{m}^3)^{-1}$. Sufficient evidence supports a mutagenic mode of action for TCE-induced kidney tumors in humans, but modes of actions have not been established for the other TCE-induced cancer types. The portion of the TCE IUR specific for kidney tumors is $1.0\text{E-}06\ (\mu\text{g}/\text{m}^3)^{-1}$, while the IUR for non-Hodgkin's lymphoma plus liver and biliary tract cancers is $3.1\text{E-}06\ (\mu\text{g}/\text{m}^3)^{-1}$.

Risk Characterization

The EPA's RCRA and Superfund programs characterize potential human health risks using standardized equations that combine toxicity values with exposure parameters because risk is a function of both hazard and exposure. Typically, the EPA's standard default exposure parameters for chronic scenarios, published in OSWER Directive 9200.1-120 (EPA, 2014b), are used. However, exposure assessments must take into account the time scale related to the specific biological response (NRC, 1991). This means that exposure parameters selected to evaluate risks and/or develop levels of concern for a given chemical and scenario should correspond as closely as possible with the exposure period used to develop the toxicity value. For example, time-weighted average exposures over a lifetime have little relevance for a developmental toxin if the adverse effects could only occur following exposure during a particular stage of development (EPA, 1992).

Non-Cancer Hazard Quotients for Cardiac Defects

The toxicity values considered protective for a lifetime of exposure to TCE are partly based on non-cancer health effects resulting from less-than-lifetime exposures. As previously stated, one of the two co-critical endpoints that serves as the basis for the TCE RfC is cardiac defects. This effect can only occur when the fetus is exposed during the period of heart development. Therefore, the EPA's standard default exposure parameters for chronic exposures are invalid for estimating hazard quotients representing the potential for cardiac defects associated with TCE exposures and for deriving TCE levels of concern that are protective of developmental endpoints. To select appropriate less-than-lifetime exposure parameters that may be used to characterize these hazards and derive levels of concern, the critical exposure period of concern for TCE-related heart malformations must first be identified.

"[F]or developmental toxic effects, a primary assumption is that a single exposure at a critical time in development may produce an adverse developmental effect, i.e., repeated exposure is not a necessary prerequisite for developmental toxicity to be manifested" (EPA, 1991). The EPA's Risk Assessment Guidance for Superfund Part A (EPA, 1989) directs the use of a day or a single exposure incident to assess the potential risks of adverse developmental effects. Following this guidance, it is assumed that a

single exposure to TCE at any time during the approximate three week period of valvuloseptal morphogenesis could result in one or more of the types of heart malformations described previously. Thus, the critical exposure period of concern used to evaluate the potential for cardiac defects is one day. A 24-hour exposure period has been used by the EPA to evaluate acute hazards associated with TCE in the final, peer-reviewed TSCA Work Plan Chemical Risk Assessment (EPA, 2014c).

The EPA's Risk Assessment Guidance for Superfund Part F, Supplemental Guidance for Inhalation Risk Assessment (EPA, 2009) specifies that the exposure concentration (EC) that should be used to evaluate risks and derive levels of concern for acute endpoints is equivalent to the concentration detected in air (CA), as shown in Equation 1.

$$EC \left(\frac{\mu g}{m^3} \right) = CA \left(\frac{\mu g}{m^3} \right) \quad (1)$$

For a residential scenario, in which exposure to TCE inside a home is assumed to occur throughout the entire exposure period of concern, Equation 1 is appropriate. However, for other types of scenarios (e.g., industrial, commercial, recreational), exposures to TCE only occur for a portion of any given 24-hour period. Moreover, exposures to different concentrations of TCE may occur within a single day at some sites. To account for these multiple exposures, Equation 1 can be modified, resulting in a time-weighted average exposure concentration. The 24-hour TWA exposure concentration can be calculated using Equation 2.

$$EC_{24} = \sum_{i=1}^n (CA_i \cdot ET_i) / AT_{24} \quad (2)$$

where: $EC_{24} (\mu g/m^3)$ = time-weighted average exposure concentration over 24 hours;
 $CA_i (\mu g/m^3)$ = TCE concentration in air in microenvironment (ME) i;
 ET_i (hours) = exposure time spent in ME i;
 AT_{24} (hours) = averaging time for the exposure period of concern (24 hours)

In a residential scenario, there is a single microenvironment, the residence, with an exposure time of 24 hours. Thus, the Residential EC_{24} will equal CA_{res} , as shown in Equation 3. To reduce uncertainty in residential scenarios, CA_{res} should be based on air samples collected for an entire 24-hour exposure period. Generally, stationary 24-hour indoor air sample results are used.

$$Residential \ EC_{24} = \frac{(CA_{res} \cdot 24 \text{ hrs})}{24 \text{ hrs}} = CA_{res} \quad (3)$$

In a typical industrial or commercial scenario, there are two microenvironments. One is the workplace, and the other is away from the workplace. The Industrial/Commercial EC_{24} can be calculated using Equation 4, below. Although the standard value for ET_{work} is an 8-hour workday, this variable should reflect site-specific conditions. For example, employees at a given site may work longer shifts, such as 10 or 12 hours, and they may or may not take their lunch breaks on site. CA_{work} should be based on air samples collected for the entire exposure time, ET_{work} , during the portion of the day that workers are present. This is to prevent potential underestimates of TCE concentrations if diurnal variations occur at a site, although such variability does not exist at all sites. Generally, stationary 8-hour or 10-hour indoor air samples are appropriate. ET_{away} should equal the remainder of the 24-hour period spent away from the workplace. CA_{away} is generally assumed to equal zero, unless site-specific data suggest otherwise.

$$Industrial/Commercial \ EC_{24} = \frac{(CA_{work} \cdot ET_{work}) + (CA_{away} \cdot ET_{away})}{24 \text{ hrs}} \quad (4)$$

If multiple or variable microenvironments are present at a site, it is possible to use Equation 2 to generate a 24-hour TWA exposure concentration. However, consideration should be given to the use of portable sampling equipment to more accurately measure true exposure concentrations to the receptor(s) of concern over the entire exposure time, as opposed to stationary sampling equipment positioned in multiple areas where exposure occurs.

Non-cancer hazard quotients for heart defects can be derived using Equation 5, where HQ_{24} is the developmental hazard quotient; EC_{24} is the 24-hr time-weighted average exposure concentration calculated using Equations 2, 3, or 4; and the RfC is $2 \mu\text{g}/\text{m}^3$. As shown in Equation 5, a hazard quotient is the ratio of the exposure to the non-cancer toxicity value. Thus, an HQ greater than 1 means that the exposure is greater than the RfC and exceeds a level of concern for that particular non-cancer health effect.

$$HQ_{24} = \frac{EC_{24}}{RfC} \quad (5)$$

Equation 5 can be combined with Equation 3 or 4 to calculate the developmental hazard quotients (HQ_{24}) for a residential or industrial/commercial receptor, as follows.

$$\text{Residential } HQ_{24} = \frac{CA_{res}}{2 \frac{\mu\text{g}}{\text{m}^3}} \quad (6)$$

$$\text{Industrial/Commercial } HQ_{24} = \frac{(CA_{work} \cdot ET_{work}) + (CA_{away} \cdot ET_{away})}{24 \text{ hrs} \cdot 2 \frac{\mu\text{g}}{\text{m}^3}} \quad (7)$$

Non-Cancer Hazard Quotients for Chronic Health Effects

Autoimmune disease, a co-critical endpoint upon which the TCE RfC is based, and kidney toxicity, the supporting endpoint, are both health effects associated with chronic or long-term exposures. Equation 8 is the standardized equation used to evaluate non-cancer hazard quotients for chronic health effects; the exposure parameters are defined in Table 1. If seasonal or temporal fluctuations in TCE concentrations potentially exist, consideration should be given as to whether sufficient data are available to generate an average concentration for use as the CA term. If the dataset is limited, it may be more health-protective to use the highest concentration detected.

$$HQ_{chronic} = \frac{CA \left(\frac{\mu\text{g}}{\text{m}^3} \right) \cdot ET \left(\frac{\text{hrs}}{\text{day}} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF \left(\frac{\text{days}}{\text{year}} \right) \cdot ED (\text{years})}{AT_{nc,chronic} (\text{days}) \cdot RfC \left(\frac{\mu\text{g}}{\text{m}^3} \right)} \quad (8)$$

The above equation can be presented in terms of residential or industrial/commercial exposure scenarios, as shown below. Note that it is only appropriate to calculate non-cancer hazard quotients for chronic health effects for those receptors with long-term exposures.

$$\text{Residential } HQ_{chronic} = \frac{CA_{res} \left(\frac{\mu\text{g}}{\text{m}^3} \right) \cdot ET_{res} \left(\frac{\text{hrs}}{\text{day}} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF_{res} \left(\frac{\text{days}}{\text{year}} \right) \cdot ED_{child} (\text{years})}{AT_{nc,chronic,child} (\text{days}) \cdot RfC \left(\frac{\mu\text{g}}{\text{m}^3} \right)} \quad (9)$$

$$\text{Industrial/Commercial } HQ_{chronic} = \frac{CA_{work} \left(\frac{\mu\text{g}}{\text{m}^3} \right) \cdot ET_{work} \left(\frac{\text{hrs}}{\text{day}} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF_{work} \left(\frac{\text{days}}{\text{year}} \right) \cdot ED_{work} (\text{years})}{AT_{nc,chronic,work} (\text{days}) \cdot RfC \left(\frac{\mu\text{g}}{\text{m}^3} \right)} \quad (10)$$

Cancer Risks

TCE is classified “carcinogenic to humans,” based on kidney cancer, non-Hodgkin’s lymphoma, and liver and biliary tract cancer. Equation 11 is the standardized equation used to evaluate excess individual lifetime cancer risks; the exposure parameters are defined in Table 1. If temporal fluctuations in TCE concentrations potentially exist, consideration should be given as to whether sufficient data are available to generate an average concentration for use as the CA term. If the dataset is limited, it may be more health-protective to use the highest concentration detected.

$$CR = \frac{CA\left(\frac{\mu g}{m^3}\right) \cdot ET\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF\left(\frac{days}{year}\right) \cdot ED(years) \cdot IUR\left(\frac{\mu g}{m^3}\right)^{-1}}{AT_{cancer}(days)} \quad (11)$$

The above equation can be presented in terms of residential or industrial/commercial exposure scenarios, as shown below. Because a mutagenic mode of action has been established for kidney tumors associated with TCE, it is necessary to apply age-dependent adjustment factors when deriving risks for this cancer type in children (EPA, 2005b). ADAFs are not applied when deriving risks for non-Hodgkin’s lymphoma or liver and biliary tract cancers associated with TCE exposures because they have not been determined to operate via a mutagenic mode of action. Because only adults are evaluated in an industrial/commercial exposure scenario and no adjustments for mutagenicity are made for adults (i.e., $ADAF_{adult} = 1$), ADAFs are not included in Equation 13.

$$\begin{aligned} \text{Residential } CR = & \left(\frac{CA_{res}\left(\frac{\mu g}{m^3}\right) \cdot ET_{res}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{res}\left(\frac{days}{year}\right)}{AT_{cancer}(days)} \right) \cdot \left[\left(ED_{0-2}(years) \cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1} \cdot \right. \right. \\ & \left. \left. ADAF_{0-2} \right) + \left(ED_{2-16}(years) \cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1} \cdot ADAF_{2-16} \right) + \left(ED_{16-26}(years) \cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1} \cdot \right. \right. \\ & \left. \left. ADAF_{adult} \right) + \left(ED_{res}(years) \cdot IUR_{N\&L}\left(\frac{\mu g}{m^3}\right)^{-1} \right) \right] \quad (12) \end{aligned}$$

$$\begin{aligned} \text{Industrial/Commercial } CR = & \frac{CA_{work}\left(\frac{\mu g}{m^3}\right) \cdot ET_{work}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{work}\left(\frac{days}{year}\right) \cdot ED_{work}(years) \cdot IUR\left(\frac{\mu g}{m^3}\right)^{-1}}{AT_{cancer}(days)} \quad (13) \end{aligned}$$

The definitions, values, and references for the exposure parameters and toxicity values used in this document are provided in Table 1. For the chronic scenarios, the EPA’s standard default exposure parameters (EPA, 2014b) are used to best represent reasonable maximum exposure scenarios, which are the highest exposures reasonably expected to occur at a site (EPA, 1989). These values are based on the 2011 Exposure Factors Handbook (EPA, 2011c). Although the default exposure time for an indoor worker is 8 hours/day, it is preferable to identify a site-specific worker exposure time.

Table 1. Exposure Parameters and Toxicity Values.

Parameter	Definition	Units	Value	Reference
$ADAF_{0-2}$	Age-dependent adjustment factor – ages 0 to 2 years	-	10	EPA, 2005b
$ADAF_{2-16}$	Age-dependent adjustment factor – ages 2 to 16 years	-	3	EPA, 2005b
$ADAF_{adult}$	Age-dependent adjustment factor – ages 16 years and older	-	1	EPA, 2005b
AT_{24}	Averaging time – developmental effects	hours	24	-
AT_{cancer}	Averaging time – cancer	days	25,550	EPA, 2014b

Table 1. Exposure Parameters and Toxicity Values.				
Parameter	Definition	Units	Value	Reference
AT _{nc,chronic, child}	Averaging time – chronic non-cancer health effects, resident child	days	2,190	EPA, 2014b
AT _{nc,chronic, work}	Averaging time – chronic non-cancer health effects, indoor worker	days	9,125	EPA, 2014b
CA	Concentration of TCE in air	µg/m ³	Measured	-
CA _{res}	Concentration of TCE in air of the residence	µg/m ³	Measured	-
CA _{work}	Concentration of TCE in air of the workplace	µg/m ³	Measured	-
ED ₀₋₂	Exposure duration – ages 0 to 2 years	years	2	EPA, 2005b
ED ₂₋₁₆	Exposure duration – ages 2 to 16 years	years	14	EPA, 2005b
ED ₁₆₋₂₆	Exposure duration – ages 16 to 26 years	years	10	EPA, 2005b
ED _{child}	Exposure duration – resident (child, ages 0 to 6 years)	years	6	EPA, 2014b
ED _{res}	Exposure duration – resident (child + adult, ages 0 to 26 years)	years	26	EPA, 2014b
ED _{work}	Exposure duration – indoor worker	years	25	EPA, 2014b
EF _{res}	Exposure frequency – resident	days/yr	350	EPA, 2014b
EF _{work}	Exposure frequency – indoor worker	days/yr	250	EPA, 2014b
ET _{away}	Exposure time – time spent away from work by an indoor worker (24 hrs/day minus ET _{work})	hrs/day	16 or site-specific	-
ET _{res}	Exposure time – time spent at home by a resident	hrs/day	24	EPA, 2014b
ET _{work}	Exposure time – time spent at work by an indoor worker	hrs/day	8 or site-specific	EPA, 2014b or site-specific
IUR	TCE inhalation unit risk - total	(µg/m ³) ⁻¹	4.1E-06	EPA, 2011a
IUR _{kid}	TCE inhalation unit risk – kidney cancer	(µg/m ³) ⁻¹	1.0E-06	EPA, 2011a
IUR _{N&L}	TCE inhalation unit risk – non-Hodgkin's lymphoma and liver and biliary tract cancers	(µg/m ³) ⁻¹	3.1E-06	EPA, 2011a
RfC	TCE reference concentration	µg/m ³	2	EPA, 2011a
THQ	Target hazard quotient	-	1	-
TR	Target cancer risk	-	1E-04	Upper-end of Target Cancer Risk Range

Action Levels

Level of Concern for Developmental Effects

Equations 2 and 5 can be manipulated to solve for the level of concern for developmental health effects, using a target non-cancer hazard quotient of 1, as follows. Note that the only exposure parameter that can vary in this calculation is the exposure time. The TCE levels of concern for developmental effects based on standard exposure times are provided in Table 2. For a 24-hour residential scenario, the developmental LOC equals 2 µg/m³. For a typical 8-hour industrial/commercial scenario, the developmental LOC equals 6 µg/m³. Site-specific developmental LOCs may be derived using alternate exposure times; for example, a 10-hour exposure time results in a developmental LOC of 4.8 µg/m³.

$$TCE\ LOC_{developmental} \left(\frac{\mu g}{m^3} \right) = \frac{THQ \cdot AT_{24}(hrs) \cdot RfC \left(\frac{\mu g}{m^3} \right)}{ET \left(\frac{hrs}{day} \right)} \quad (14)$$

$$TCE\ Residential\ LOC_{developmental} \left(\frac{\mu g}{m^3} \right) = \frac{1 \cdot 24\ hrs \cdot 2 \frac{\mu g}{m^3}}{24\ hrs} \quad (15)$$

$$TCE \text{ Industrial/Commercial } LOC_{developmental} \left(\frac{\mu g}{m^3} \right) = \frac{1 \cdot 24 \text{ hrs} \cdot 2 \frac{\mu g}{m^3}}{ET_{work} \left(\frac{hrs}{day} \right)} \quad (16)$$

Level of Concern for Chronic Non-Cancer Health Effects

Equation 8 can be manipulated to solve for the level of concern for chronic, non-cancer health effects, using a target non-cancer hazard quotient of 1 and the exposure parameters presented in Table 1, as follows. For a residential scenario, this LOC equals $2.1 \mu g/m^3$, which is the value listed as the non-cancer residential air Regional Screening Level for TCE, based on an HQ of 1 (EPA, 2016b). For an industrial/commercial scenario, the chronic LOC equals $8.8 \mu g/m^3$, which is the value listed as the non-cancer worker air RSL for TCE, based on an HQ of 1. Site-specific chronic LOCs may be derived using alternate exposure times or other parameters.

$$TCE \text{ } LOC_{chronic} \left(\frac{\mu g}{m^3} \right) = \frac{THQ \cdot AT_{nc,chronic}(days) \cdot Rfc \left(\frac{\mu g}{m^3} \right)}{ET \left(\frac{hrs}{day} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF \left(\frac{days}{year} \right) \cdot ED(years)} \quad (17)$$

$$TCE \text{ Residential } LOC_{chronic} \left(\frac{\mu g}{m^3} \right) = \frac{THQ \cdot AT_{nc,chronic,child}(days) \cdot Rfc \left(\frac{\mu g}{m^3} \right)}{ET_{res} \left(\frac{hrs}{day} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF_{res} \left(\frac{days}{year} \right) \cdot ED_{child}(years)} \quad (18)$$

$$TCE \text{ Industrial/Commercial } LOC_{chronic} \left(\frac{\mu g}{m^3} \right) = \frac{THQ \cdot AT_{nc,chronic,work}(days) \cdot Rfc \left(\frac{\mu g}{m^3} \right)}{ET_{work} \left(\frac{hrs}{day} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF_{work} \left(\frac{days}{year} \right) \cdot ED_{work}(years)} \quad (19)$$

Level of Concern for Cancer Risks

Equations 11, 12, and 13 can be manipulated to solve for the level of concern for cancer risks, using a target excess cancer risk (TR) of $1E-04$, which is the upper bound of the EPA's target cancer risk range, and the exposure parameters presented in Table 1, as follows. For a residential scenario, this LOC equals $48 \mu g/m^3$, and for an industrial/commercial scenario, the cancer LOC equals $300 \mu g/m^3$.

$$TCE \text{ } LOC_{cancer} \left(\frac{\mu g}{m^3} \right) = \frac{TR \cdot AT_{cancer}(days)}{ET \left(\frac{hrs}{day} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF \left(\frac{days}{year} \right) \cdot ED(years) \cdot IUR \left(\frac{\mu g}{m^3} \right)^{-1}} \quad (20)$$

$$TCE \text{ Residential } LOC_{cancer} \left(\frac{\mu g}{m^3} \right) = \frac{TR \cdot AT_{cancer}(days)}{ET \left(\frac{hrs}{day} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF \left(\frac{days}{year} \right) \cdot \left[\left(ED_{0-2}(years) \cdot IUR_{kid} \left(\frac{\mu g}{m^3} \right)^{-1} \cdot ADAF_{0-2} \right) + \left(ED_{2-16}(years) \cdot IUR_{kid} \left(\frac{\mu g}{m^3} \right)^{-1} \cdot ADAF_{2-16} \right) + \left(ED_{16-26}(years) \cdot IUR_{kid} \left(\frac{\mu g}{m^3} \right)^{-1} \cdot ADAF_{16-26} \right) + \left(ED_{res}(years) \cdot IUR_{N\&L} \left(\frac{\mu g}{m^3} \right)^{-1} \right) \right]} \quad (21)$$

$$TCE \text{ Industrial/Comm. } LOC_{cancer} \left(\frac{\mu g}{m^3} \right) = \frac{TR \cdot AT_{cancer}(days)}{ET_{work} \left(\frac{hrs}{day} \right) \cdot \left(\frac{1 \text{ day}}{24 \text{ hrs}} \right) \cdot EF_{work} \left(\frac{days}{year} \right) \cdot ED_{work}(years) \cdot IUR \left(\frac{\mu g}{m^3} \right)^{-1}} \quad (22)$$

As shown below in Table 2, the levels of concern for developmental health effects are lower than the LOCs for chronic health effects and cancer, for both residential and occupational scenarios, when based on target hazard quotients of 1 or target cancer risks of $1E-04$. These are the levels of risk that, when exceeded, warrant action under the National Contingency Plan. Basing the Region 7 TCE action levels

on the developmental LOCs is protective for all potential forms of adverse health effects associated with TCE. Thus, the action level for a residential scenario is 2 $\mu\text{g}/\text{m}^3$, and the action level for a typical industrial/commercial scenario with an 8-hr workday is 6 $\mu\text{g}/\text{m}^3$. As previously mentioned, the developmental LOC, and thus the action level, is highly dependent on the exposure time. Therefore, for non-residential exposure scenarios, careful consideration should be given to the value selected as the exposure time.

Table 2. Levels of Health Concern for Trichloroethylene ($\mu\text{g}/\text{m}^3$), THQ = 1 and TR = 1E-04.	
Residents (24-hr Exposure Scenario)	
<i>Developmental Non-Cancer LOC:</i>	2
<i>Chronic Non-Cancer LOC:</i>	2.1
<i>Cancer LOC:</i>	48
Region 7 Residential TCE Action Level:	2
Industrial/Commercial Workers (8-hr Exposure Scenario)	
<i>Developmental Non-Cancer LOC:</i>	6
<i>Chronic Non-Cancer LOC:</i>	8.8
<i>Cancer LOC:</i>	300
Region 7 Industrial/Commercial TCE Action Level:	6

Risk Management Considerations

If the TCE action level is exceeded, this indicates a potential imminent threat to human health, and early or interim action(s) should be taken to eliminate, reduce, and/or control the hazards posed by the site (EPA, 2014d). At Superfund sites, coordination between the remedial and removal programs should immediately commence as early as the receipt of preliminary sampling results indicative of a potential human health concern (EPA, 2016c). Potential receptors should be informed of the results and potential risks to human health. Standard Region 7 practice is to communicate this information via data transmittal letters submitted to property owners and employers, but when TCE action levels are exceeded, tenants, residents, employees and others who may be exposed should also be informed. Although the action levels derived in this document are applicable to women in the first trimester of pregnancy, note that the levels protective of autoimmune disease and kidney toxicity in all individuals are not significantly different, at 2.1 and 8.8 $\mu\text{g}/\text{m}^3$, for residents and workers, respectively. Depending on the concentrations detected, immediate site actions could include relocation, restricting the time residents or workers remain in areas exceeding action levels, opening basement or lower level windows for ventilation (using a fan), sealing cracks in the slab, sealing sump pits, sealing cinder block or stone walls, and/or using air filtration systems. Vapor mitigation systems or adjustments to HVAC systems may be used to minimize exposures on a more long-term basis. Post-remedy testing and continued operation and maintenance is necessary to ensure protection of human health until the source of TCE in soil and/or groundwater is ultimately addressed.

Other EPA Regions and states have derived tiered action levels prescribing the types and urgency of various responses, as described below.

- Although Region 7 consistently uses a THQ of 1 as the basis for both removal and remedial Superfund actions, other Regions have used a THQ of 3 as a science policy approach to prioritize actions that may warrant the use of removal authority, with ultimate cleanup goals based on a THQ of 1. Since non-cancer toxicity values have historically been based on effects resulting from chronic exposure, this practice assumes that the most highly contaminated sites will be remediated first, but all sites will be remediated before exposures have occurred for a sufficiently long duration (e.g., 25 years as a worker or 26 years as a resident) to pose significant health risks.

This assumption is not protective of the short-term health effects associated with TCE, in which the critical window of susceptibility is an approximate three week period and a single exposure during this critical time may result in cardiac malformations.

- Tiered action levels could also be derived by reducing the uncertainty factor applied to the RfC from 10 to 1. The existing UF of 10 is applied for uncertainty regarding differences in pharmacodynamics between animals and humans and between the general population and sensitive subpopulation. Other than the toxicokinetic variability characterized by the physiologically-based pharmacokinetic model, EPA (2011a) indicates that there are inadequate chemical-specific data to quantify the degree of differential susceptibility due to factors such as genetic polymorphisms, race/ethnicity, preexisting health status, lifestyle factors, and nutritional status. The UF of 10 was included in the extensive peer-review process described in this document, and Region 7 does not have justification to alter this value.
- Similarly, the selection of a 1% excess risk as the benchmark response and a human equivalent concentration for a toxicokinetically sensitive individual at the 99th percentile were both extensively reviewed, and Region 7 does not have justification to alter these criteria.

Although Region 7 has not developed tiered levels because this approach may not be protective of human health, higher concentrations of TCE are associated with greater health risks. Actions should be implemented as quickly as is practicable to minimize risks of developmental toxicity. This document reinforces that Region 7 should expedite actions to protect human health whenever the TCE air concentration exceeds $2 \mu\text{g}/\text{m}^3$ in a residential scenario or $6 \mu\text{g}/\text{m}^3$ for an 8-hour worker scenario.

REFERENCES

- Dhanantwari, P; Lee, E; Krishnan, A; Samtani, R; Yamada, S; Anderson, S; Locket, E; Donofrio, M; Shiota, K; Leatherbury, L; Lo, CW. 2009. Human cardiac development in the first trimester: a high-resolution magnetic resonance imaging and episcopic fluorescence image capture atlas. *Circulation* 120: 343–51.
- Halogenated Solvents Industry Alliance, Inc. 2013. Request for Correction – IRIS Assessment for Trichloroethylene. RFC 14001. November 5, 2013.
- Halogenated Solvents Industry Alliance, Inc. 2015. Request for Reconsideration/#14001. RFR 14001A. June 17, 2015.
- Johnson, P.; Goldberg, S.; Mays, M.; Dawson, B. 2003. Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat. *Environ Health Perspect.* 111: 289-292. <http://www.ncbi.nlm.nih.gov/pubmed/12611656>
- Keil, D; Peden-Adams, M; Wallace, S; Ruiz, P; Gilkeson, G. 2009. Assessment of trichloroethylene (TCE) exposure in murine strains genetically-prone and non-prone to develop autoimmune disease. *J Environ Sci Health A Tox Hazard Subst Environ Eng.* 44: 443-453. <http://dx.doi.org/10.1080/10934520902719738>
- Makris, SL; Scott, CS; Fox, J; Knudsen, TB; Hotchkiss, AK; Arzuaga, X; Euling, SY; Powers, CM; Jinot, J; Hogan, KA; Abbott, BD; Hunter III, ES; Narotsky, MG. 2016. A systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development. *Reprod Toxicol.* 65(2016): 321-358.
- National Aeronautics and Space Administration. 2009. NASA Review/Comments on Draft EPA TCE IRIS Risk Assessment. August 26, 2009. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=492690
- National Aeronautics and Space Administration. 2011. NASA Comments on the Final Draft TCE IRIS Risk Assessment. July 2011. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=503452
- National Research Council. 1991. Human Exposure Assessment for Airborne Pollutants: Advances and Opportunities. Washington, DC: The National Academy Press, 1991.
- National Research Council. 2006. Assessing the Human Health Risks of Trichloroethylene: Key Scientific Issues. Washington, DC: The National Academies Press, 2006.
- Office of Management and Budget. 2009. OMB Staff Comments on EPA IRIS draft TCE Toxicological Review and Draft Charge (drafts dated June 2009). September, 1, 2009. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=492688
- Office of Management and Budget. 2011. OMB Staff Working Comments on EPA's Final Agency/Interagency Science Discussion draft Toxicological Review of Trichloroethylene (TCE) and draft IRIS Summary (dated June 2011). July 21, 2011. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=503454

- U.S. Department of Defense. 2009a. Department of Defense Comments on the Draft TCE Toxicological Review; Peer Review Charge, June 2009. The Chemical Material Risk Management Directorate. August 27, 2009. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=492692
- U.S. Department of Defense. 2009b. Department of Defense Comments on the IAR Draft USEPA TCE Risk Assessment, June 2009. The Chemical Material Risk Management Directorate. September 1, 2009. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=492691
- U.S. Department of Defense. 2011. Department of Defense Comments on TCE IASD final draft Tox Review redline.pdf. The Chemical Material Risk Management Directorate. July 15, 2011. http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=503451
- U.S. Environmental Protection Agency. 1989. Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual - Part A. EPA/540/1-89/002. Office of Emergency and Remedial Response, Washington, D.C.
- U.S. Environmental Protection Agency. 1991. Guidelines for Developmental Toxicity Risk Assessment. EPA/600/FR-91/001. Risk Assessment Forum, Washington, D.C.
- U.S. Environmental Protection Agency. 1992. Guidelines for Exposure Assessment. EPA/600/Z-92/001. Risk Assessment Forum, Washington, D.C.
- U.S. Environmental Protection Agency. 2002. Review of Draft Trichloroethylene Health Risk Assessment: Synthesis and Characterization: An EPA Science Advisory Board Report. EPA-SAB-EHC-03-002. TCE Review Panel, Environmental Health Committee, US EPA Science Advisory Board. Washington, D.C. December 2002. [http://yosemite.epa.gov/sab/sabproduct.nsf/D14C306CF5482E41852571CE00697543/\\$File/ehc03002.pdf](http://yosemite.epa.gov/sab/sabproduct.nsf/D14C306CF5482E41852571CE00697543/$File/ehc03002.pdf)
- U.S. Environmental Protection Agency. 2003. Human Health Toxicity Values in Superfund Risk Assessments. OSWER Directive 9285.7-53. Office of Superfund Remediation and Technology Innovation, Washington, D.C.
- U.S. Environmental Protection Agency. 2005a. Guidelines for Carcinogen Risk Assessment. EPA/630/P-03/001F. Risk Assessment Forum, Washington, D.C.
- U.S. Environmental Protection Agency. 2005b. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. EPA/630/R-03/003F. Risk Assessment Forum, Washington, D.C.
- U.S. Environmental Protection Agency. 2009. Risk Assessment Guidance for Superfund: Volume I – Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment). OSWER Publication 9285.7-82. Office of Superfund Remediation and Technology Innovation, Washington, D.C.
- U.S. Environmental Protection Agency. 2011a. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). EPA/635/R-09/011F. Washington, D.C. September 2011.

- U.S. Environmental Protection Agency. 2011b. Review of EPA's Draft Assessment entitled 'Toxicological Review of Trichloroethylene' (October 2009). EPA-SAB-11-002. TCE Review Panel, US EPA Science Advisory Board. Washington, D.C. January 11, 2011.
[http://yosemite.epa.gov/sab%5Csabproduct.nsf/B73D5D39A8F184BD85257817004A1988/\\$File/EPA-SAB-11-002-unsigned.pdf](http://yosemite.epa.gov/sab%5Csabproduct.nsf/B73D5D39A8F184BD85257817004A1988/$File/EPA-SAB-11-002-unsigned.pdf)
- U.S. Environmental Protection Agency. 2011c. Exposure Factors Handbook: 2011 edition. National Center for Environmental Assessment, Washington, DC; EPA/600/R-09/052F.
- U.S. Environmental Protection Agency. 2014a. TCE Developmental Cardiac Toxicity Assessment Update. EPA-HQ-OPPT-2012-0723-0045. July 2014.
<http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPPT-2012-0723-0045>
- U.S. Environmental Protection Agency. 2014b. Human Health Evaluation Manual, Supplemental Guidance: Update of Standard Default Exposure Factors. OSWER Directive 9200.1-120. Office of Solid Waste and Emergency Response, Washington, D.C.
- U.S. Environmental Protection Agency. 2014c. TSCA Work Plan Chemical Risk Assessment. Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses. CASRN: 79-01-6. EPA Document 740-R1-4002. Office of Chemical Safety and Pollution Prevention, Washington, D.C. June 2014
- U.S. Environmental Protection Agency. 2014d. Compilation of Information Relating to Early/Interim Actions at Superfund Sites and the TCE IRIS Assessment. OSWER Directive 9200.1-120. Office of Solid Waste and Emergency Response, Washington, D.C. August 27, 2014.
- U.S. Environmental Protection Agency. 2015. EPA Response to RFC 14001. March 19, 2015.
- U.S. Environmental Protection Agency. 2016a. EPA Response to RFR 14001A. February 26, 2016.
- U.S. Environmental Protection Agency. 2016b. Regional Screening Levels for Chemical Contaminants at Superfund Sites. Accessed September 22, 2016. <https://www.epa.gov/risk/regional-screening-levels-rsls>
- U.S. Environmental Protection Agency. 2016c. Remedial and Removal Program Coordination Regarding Potential Imminent Threats to Human Health or the Environment. Office of Land and Emergency Management, Washington, D.C. August 11, 2016.

**FANTASTIC CAVERNS
GREENE COUNTY
*EMPLOYEE FACT SHEET - TCE EXPOSURES 2016***



BACKGROUND

- ◆ Trichloroethylene (TCE), a chemical historically used as a solvent to clean metal parts and which readily evaporates into air, has been found in the air within Fantastic Caverns. This sampling was conducted as part of an environmental investigation to determine if air inside caves visited by the public is affected by nearby sites where releases of contaminants are known to have occurred.
- ◆ The Missouri Department of Natural Resources (MoDNR) collected air samples from inside the cave in April and July 2016. TCE was detected in July above health-based levels of concern.
- ◆ The TCE in Fantastic Caverns is believed to be related to a former manufacturing facility located in Springfield, three miles south-south east of the cave. It is believed that TCE from this site has moved through soil and groundwater and eventually evaporated into the cave's air.

FUTURE SAMPLING ACTIVITIES

- ◆ Many sampling events over time are necessary to determine the variability in TCE levels due to seasonal variation in temperature, barometric pressure and air flow.
- ◆ MoDNR is discussing with the cave owner about conducting air sampling in the cave over time to evaluate TCE levels and to collect the necessary data for health officials to evaluate potential risk to employees and to determine appropriate response actions.

HEALTH INFORMATION

- ◆ In general, exposure to a chemical does not necessarily mean health effects will occur. The effect of exposure to any chemical depends on several things, including the concentration and toxicity of the chemical, the length of the exposure, and personal factors such individual health and sensitivity.
- ◆ The effect of concern from short-term exposure is the potential for fetal heart defects to occur in developing fetuses of pregnant workers, if exposure occurs during fetal heart development in the first trimester of pregnancy (weeks six through nine of gestation). Given this, there is also a concern for women of child-bearing age in general, since fetal heart development may occur during a time when a woman may not know she is pregnant.
- ◆ Workers with long-term exposure to TCE in cave air may be at increased risk of other harmful effects and possible increased cancer risks, if exposure occurs over extended periods of time. Long-term TCE exposure is associated with harmful effects to the immune system, nervous system, kidney, liver, and male reproductive system and is also associated with increased cancer risk for liver, kidney and non-Hodgkin's lymphoma.

CONTACT INFORMATION

- ◆ For health-related questions regarding TCE exposure, please contact Michelle Hartman with MDHSS at 573-751-6102.
- ◆ For information on the cave investigation, please contact Wane Roberts with MoDNR at 573-751-4187.

This fact sheet answers the most frequently asked health questions (FAQs) about trichloroethylene. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Trichloroethylene is used as a solvent for cleaning metal parts. Exposure to very high concentrations of trichloroethylene can cause dizziness, headaches, sleepiness, incoordination, confusion, nausea, unconsciousness, and even death. The Environmental Protection Agency (EPA) and the International Agency for Research on Cancer (IARC) classify trichloroethylene as a human carcinogen. Trichloroethylene has been found in at least 1,045 of the 1,699 National Priorities List sites identified by the EPA.

What is trichloroethylene?

Trichloroethylene is a colorless, volatile liquid. Liquid trichloroethylene evaporates quickly into the air. It is nonflammable and has a sweet odor.

The two major uses of trichloroethylene are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a. Trichloroethylene was once used as an anesthetic for surgery.

What happens to trichloroethylene when it enters the environment?

- Trichloroethylene can be released to air, water, and soil at places where it is produced or used.
- Trichloroethylene is broken down quickly in air.
- Trichloroethylene breaks down very slowly in soil and water and is removed mostly through evaporation to air.
- It is expected to remain in groundwater for long time since it is not able to evaporate.
- Trichloroethylene does not build up significantly in plants or animals.

How might I be exposed to trichloroethylene?

- Breathing trichloroethylene in contaminated air.
- Drinking contaminated water.
- Workers at facilities using this substance for metal degreasing are exposed to higher levels of trichloroethylene.
- If you live near such a facility or near a hazardous waste site containing trichloroethylene, you may also have higher exposure to this substance.

How can trichloroethylene affect my health?

Exposure to moderate amounts of trichloroethylene may cause headaches, dizziness, and sleepiness; large amounts may cause coma and even death. Eating or breathing high levels of trichloroethylene may damage some of the nerves in the face. Exposure to high levels can also result in changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Skin contact with concentrated solutions of trichloroethylene can cause skin rashes.

There is some evidence exposure to trichloroethylene in the work place may cause scleroderma (a systemic autoimmune disease) in some people. Some men occupationally-exposed to trichloroethylene and other chemicals showed decreases in sex drive, sperm quality, and reproductive hormone levels.

How likely is trichloroethylene to cause cancer?

There is strong evidence that trichloroethylene can cause kidney cancer in people and some evidence for trichloroethylene-induced liver cancer and malignant lymphoma. Lifetime exposure to trichloroethylene resulted in increased liver cancer in mice and increased kidney cancer and testicular cancer in rats.

The IARC and the EPA determined that there is convincing evidence that trichloroethylene exposure can cause kidney cancer. The National Toxicology Program (NTP) is recommending a change in cancer classification to "known human carcinogen" http://ntp.niehs.nih.gov/ntp/roc/monographs/finaltce_508.pdf.

Trichloroethylene

CAS # 79-01-6

How can trichloroethylene affect children?

It is not known whether children are more susceptible than adults to the effects of trichloroethylene.

Some human studies indicate that trichloroethylene may cause developmental effects such as spontaneous abortion, congenital heart defects, central nervous system defects, and small birth weight. However, these people were exposed to other chemicals as well.

In some animal studies, exposure to trichloroethylene during development caused decreases in body weight, increases in heart defects, changes to the developing nervous system, and effects on the immune system.

How can families reduce the risk of exposure to trichloroethylene?

- Avoid drinking water from sources that are known to be contaminated with trichloroethylene. Use bottled water if you have concerns about the presence of chemicals in your tap water. You may also contact local drinking water authorities and follow their advice.
- Discourage your children from putting objects in their mouths. Make sure that they wash their hands frequently and before eating.
- Prevent children from playing in dirt or eating dirt if you live near a waste site that has trichloroethylene.
- Trichloroethylene is used in many industrial products. Follow instructions on product labels to minimize exposure to trichloroethylene.

Is there a medical test to show whether I've been exposed to trichloroethylene?

Trichloroethylene and its breakdown products (metabolites) can be measured in blood and urine. However, the detection of trichloroethylene or its metabolites cannot predict the kind of health effects that might develop from that exposure. Because trichloroethylene and its metabolites leave the body fairly rapidly, the tests need to be conducted within days after exposure.

Has the federal government made recommendations to protect human health?

The EPA set a maximum contaminant goal (MCL) of 0.005 milligrams per liter (mg/L; 5 ppb) as a national primary drinking standard for trichloroethylene.

The Occupational Safety and Health Administration (OSHA) set a permissible exposure limit (PEL) of 100 ppm for trichloroethylene in air averaged over an 8-hour work day, an acceptable ceiling concentration of 200 ppm provided the 8 hour PEL is not exceeded, and an acceptable maximum peak of 300 ppm for a maximum duration of 5 minutes in any 2 hours.

The National Institute for Occupational Safety and Health (NIOSH) considers trichloroethylene to be a potential occupational carcinogen and established a recommended exposure limit (REL) of 2 ppm (as a 60-minute ceiling) during its use as an anesthetic agent and 25 ppm (as a 10-hour TWA) during all other exposures.

References

This ToxFAQs™ information is taken from the 2014 Toxicological Profile for Trichloroethylene (Draft for Public Comment) produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30329-4027.

Phone: 1-800-232-4636.

ToxFAQs™ on the web: www.atsdr.cdc.gov/toxFAQs.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.